Fluid Shifts and Hydration State: Effects of Long-Term Exercise

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Catalogue Data:
CONVERTINO, V.A. (1987). Fluid shifts and hydration state: effects of long-term exercise. **Can. J. Spt. Sci.** 12(Suppl.1): 000S-000S.

ABSTRACT

During exercise, increased capillary hydrostatic pressure caused by elevation of arterial pressure produces plasma volume shifts from the vascular space to the interstitial fluids. Following a rapid efflux of vascular fluid within minutes of exercise, there is very little further reduction in plasma volume during long-term exercise, suggesting protective mechanisms against loss of circulating blood volume. These mechanisms probably include increasing plasma protein oncotic pressure, differences in peripheral vasoconstriction in active muscles and inactive tissues, and elevated lymph flow. The interaction of these factors provides optimal thermoregulatory and cardiovascular stability. The dynamics of fluid shifts during long-term exercise are altered by hydration state. The hypovolemia caused by dehydration acts to conserve blood volume by reducing the amount of plasma shift and sweat loss during exercise. The consequence is less heat dissipation and greater cardiovascular stability. In contrast, the hypervolemia produced by hyperhydration promotes greater shifts of fluid and sweat loss, resulting in lower body temperature and heart rate during prolonged work. The beneficial effects of hyperhydration and subsequent hypervolemia are manifest in the adaptation of body fluids and electrolytes to exercise training. Thus, with regard to fluid shifts during long-term exercise, training is an effective way to become hyperhydrated and to reduce the limiting effects of working in 'hostile' environments.

Key words: hypervolemia, hypovolemia, plasma volume.

RÉSUMÉ

Il se produit au cours de l'exercice un déplacement du volume plasmatique à partir de l'espace vasculaire vers l'espace interstitiel en raison de l'augmentation de la pression hydrostatique capillaire consécutive à l'élévation de la pression artérielle. Lors d'un exercice prolongé, un déplacement rapide du volume vasculaire vers l'espace interstitiel survient dès les premières minutes d'exercice, qui n'est plus modifié par la suite, ce qui suggère la présence de mécanismes de contôle visant à minimiser la perte du volume sanguin circulant. Ces mécanismes comprennent probablement: l'augmentation de la pression oncotique du plasma; les différences dans le tonus vasomoteur des musles actifs ou inactifs; et l'augmentation de la circulation lymphatique. L'interaction de ces facteurs permet de constituer des conditions optimales pour la stabilité thermorégulatoire et cardiovasculaire. Les déplacements volémique au cours de l'exercice sont modifiés par l'état d'hydratation. En présence d'une hypovolémie consécutive à la déshydratation, le volume sanguin est maintenu grâce à une diminution du déplacement de volume plasmatique et une réduction de la sudation au cours de l'exercice. Ces ajustements permettront une plus grande stabilité cardiovasculaire en dépit d'une moins bonne élimination de la chaleur. Au contraire, en présence d'une hypervolémie consécutive à une hyperhydratation, un plus grand déplacement vasculaire sera observé ainsi qu'une sudation plus abondante conduisant ainsi à une température corporelle mois élevée et une fréquence cardiaque plus basse au cours de l'effort prolongé. Les effets positifs de l'hyperhydratation et de l'hypervolémie semblent evidents si l'on considère que ces manifestations sont les manifestations propres de l'entraînement physique. Ainsi, l'entraînement physique représente une stratégie d'adaptation visant à minimiser les effets limitants de l'exercice en milieu "hostile."

Mots-clés: hypervolémie, hypovolémie, volume asthmatique.

Introduction

The topics discussed at this symposium emphasize how alterations in ambient pressure, temperature and gravity can provide 'hostile' environments which significantly affect man's capacity to perform physical work. Fluid shifts induced by acute exercise and alterations in hydration state are common features to all environments and can become limiting factors to the performance of long-term exercise in heat (Harrison, 1985; Senay and Kok, 1976a; Senay *et al.*, 1976b) and at altitude (Van Beaumont *et al.*, 1971). Expansion of plasma volume following exercise training is associated with increased orthostatic tolerance (Convertino *et al.*, 1984a), and the loss of plasma volume during weightlessness reduces orthostatic and exercise tolerance upon return to the 1G environment of earth (Convertino, 1986).

The inherent physiological paradox with regard to the dynamics

of body fluid balance during exercise is the need to maintain adequate circulating fluid in the vascular space for appropriate blood flow distribution to working muscles, while promoting increased skin blood flow and fluid loss through sweating for heat dissipation. The effects of dehydration during long-term exercise can result in cardiovascular and thermoregulatory instability because of the competition for body fluids between circulation and thermoregulation. Subsequently, work performance becomes compromised by making the environment more hostile. Understanding the characteristics and mechanisms of fluid shifts during long-term exercise and the effects of varying hydration states can provide a basis for the development of protective measures against the limiting effects of these changes.

Characteristics and Mechanisms of Fluid Shifts

The dynamics of fluid shifts during exercise are characterized by a rapid movement of fluid and electrolytes from the vascular to interstitial spaces (Convertino *et al.*, 1981). This shift occurs within minutes of exercise without any change in body water content (Convertino *et al.*, 1918; Harrison, 1985) and its magnitude is dependent upon work intensity (Convertino *et al.*, 1981; Convertino *et al.*, 1983b; Wilkerson *et al.*, 1977). Since most electrolytes shift with the plasma water, any increase in electrolyte concentration and osmolality results from a more rapid movement of water than solute, resulting in a net efflux of hypotonic fluid from the vascular space (Convertino *et al.*, 1981; Van Beaumont *et al.*, 1972).

Fluid shifts from intravascular to extravascular spaces are dictated by changes in hydrostatic and oncotic pressure gradients across the capillary membranes, and the capillary membrane permeability coefficient. The precise mechanisms and magnitude of alterations in Starling forces in various tissues during exercise are unclear, since the methodology required for simultaneous measurement of all forces is not available. Thus, our knowledge of the mechanisms of fluid shifts during exercise has been limited to systemic responses based on microhematocrit measurements. The primary mechanism for the fluid shift out of the vascular space during exercise appears to be an increased arterial and capillary pressure, since there is very little net plasma protein efflux and the protein concentration increases proportionately with plasma loss (Convertino *et al.*, 1981; Harrison and Edwards, 1976; Harrison *et al.*, 1975).

Following the inital efflux of vascular fluids during exercise, the plasma volume stabilizes so that the percent loss of plasma following 30–60 minutes of exercise is similar to that after 10 minutes (Fortney *et al.*, 1981; Shaffrath and Adams, 1984; Pivarnik and Senay, 1986). These fluid dynamics suggest that there are mechanisms which protect circulating plasma volume at given levels of work. One such mechanism is the increase in plasma oncotic pressure in the capillary as a result of increasing plasma protein concentration. In addition, elevations of plasma vasopressin and renin-angiotensin are induced by exercise, and plasma levels are related to exercise intensity (Convertino *et al.*, 1981; Convertino *et al.*, 1983b). These hormone systems contribute to renal and sweat gland retention of fluids and electrolytes during exercise (Convertino and Kirby, 1985; Kirby and Convertino, 1986), although the magnitude of fluid conservation during exercise by these mechanisms may be relatively small. A more likely immediate contribution that may be made by these endocrine systems to help protect plasma volume during a long-term exercise bout could be their effect on peripheral vascular tone, since vasopressin and angiotensin are potent vasoconstrictors. A vasoconstriction in inactive tissues results in decreased mean capillary hydrostatic pressure, that would result in a greater net reabsorption of fluid into the vascular compartment from the interstitial spaces. During long-term exercise, this effect can act to replace some of the plasma volume lost by net filtration of fluid out of the vascular compartment due to local vasodilation in the active muscles and skin. In addition, return of fluid from elevated lymph flow in contracting muscle can contribute to the maintenance of plasma volume during long-term exercise (Jacobsson and Kjellmer, 1964). The net result is a dynamic fluid shift which stabilizes plasma volume reduction throughout the exercise period at a level proportionate to exercise intensity.

Thus, the actions of counterbalancing mechanisms for fluid shifts into the circulation in inactive tissue, and out of the circulation in active muscle and skin during long-term exercise allow for optimal blood volume and cardiovascular stability. It has been suggested that the fluid lost in sweat, which is obtained from the interstitial fluid surrounding the sweat glands, can be replenished by filtration of vascular fluid across local capillaries (Fortney *et al.*, 1981). However, the amount of fluid shift out of the vascular space during long-term exercise is much smaller than the total sweat loss. The most likely explanation for the fluid shift induced by exercise is the simple physical effect of altering the balance of hydrostatic and oncotic pressures in the capillaries, as described by the Starling forces. The effects of elevated plasma proteins and volume-regulating hormones act as counterbalances for the net increase in capillary hydrostatic pressure, to conserve circulating plasma and blood volume at a level which does not compromise cardiovascular effectiveness.

Effect of Hydration State

It is well documented that hydration state modifies the capillary exchange dynamics, and thus the direction and magnitude of vascular fluid changes associated with exercise (Costill and Sparks, 1973; Claremont *et al.*, 1976; Gaebelein and Senay, 1982; Sawka *et al.*, 1984). The loss of plasma volume during exercise is related to absolute blood volume. During hypovolemia following dehydration, when it is most critical to conserve blood volume to maintain cardiac filling pressure, there is a smaller loss of plasma volume than during normohydration (Fortney *et al.*, 1981; Greenleaf and Castle, 1971), higher core temperature (Candas *et al.*, 1986; Fortney *et al.*, 1981; Greenleaf and Castle, 1971), lower stroke volume (Fortney *et al.*, 1983), and higher heart rate (Candas *et al.*, 1986; Greenleaf and Castle, 1971). A greater loss of plasma occurs during hypervolemia (Fortney *et al.*, 1981), when there is a greater vascular fluid reserve. When compared to the normovolemic state, hypervolemia results in lower core temperature (Fortney *et al.*, 1981; Greenleaf and Castle, 1971), greater stroke volume (Fortney *et al.*, 1983), and lower heart rate (Candas *et al.*, 1986; Greenleaf and Castle, 1971). Therefore, the capacity to shift fluid and maintain the integrity of the cardiovascular and thermoregulatory systems during prolonged exer-

cise is dependent upon the vascular volume before exercise.

The contribution of mechanisms controlling fluid shifts during long-term exercise may differ in conditions of hyperhydration and dehydration. In the hypovolemic state, plasma protein concentration is significantly elevated above control values during rest and exercise (Candas *et al.*, 1986; Fortney *et al.*, 1981). Higher protein concentration provides a greater oncotic pressure and acts to retain fluid within the vascular compartment, resulting in a smaller net loss of plasma volume during exercise. Reduced oncotic pressure during hyperhydration may contribute somewhat to larger plasma volume loss during exercise, since plasma protein concentration is decreased (Candas *et al.*, 1986). However, greater vascular fluid shifts with hypervolemia compared to normovolemia cannot necessarily be explained by differences in oncotic pressure, since plasma protein concentrations may not differ between hyperhydration and normohydration states (Fortney *et al.*, 1981). A more likely explanation for the primary contributing factor to greater fluid shifts observed in hyperhydration may be variations that occur in peripheral vascular tone. A relatively greater vasodilation occurs in higher compared to lower volemic states (Nadel *et al.*, 1980). A greater vasodilation in inactive tissues would increase mean capillary hydrostatic pressure and promote greater net vascular fluid efflux into the interstitial space. The mechanism by which peripheral vascular tone may be less in hyperhydration and more in dehydration is not known. One possible explanation may be greater and lesser activation of vasoactive fluid-regulating hormones during hypovolemia and hypervolemia, respectively. In a recent study, Brandenberger *et al.*, (1986) demonstrated that the plasma volume loss and subsequent increase in vasopressin and renin-angiotensin during four hours of exercise were prevented by progressive rehydration. Thus, while greater capillary oncotic pressure may be the primary contributing factor to restricting fluid shifts in dehydration, greater peripheral vasodilation associated with lower levels of vasoactive hormones appears to be the primary mechanism by which greater plasma volume shifts occur in hyperhydration compared to a less hydrated state.

Although fluid shifts alone do not appear to be a limiting factor to the performance of long-term exercise, hypovolemia induced by dehydration greatly reduced the magnitude of fluid shifts compared to the hyperhydration state, thus producing greater cardiovascular and thermoregulatory limitations. Although restricted fluid shifts associated with dehydration may ultimately limit exercise performance, the subsequent hypovolemia activates mechanisms to conserve body fluids and prevent physiological collapse. The mechanisms responsible for lesser fluid shifts during dehydration appear to be a combination of protein oncotic factors and difference in control of peripheral vasoconstriction. In any case, the degree to which the environment is 'hostile' for the performance of exercise can be significantly altered by hydration state.

Adaptation to Exercise Training

Endurance training induces expansion of resting plasma volume and total circulating electrolytes and proteins (Convertino *et al.*, 1980a; Convertino *et al.*, 1980b). Endurance training also results in modification of vascular volume dynamics that is manifested by greater cardiovascular and thermoregulatory stability during acute exercise (Convertino *et al.*, 1983a; Senay, 1979; Senay *et al.*, 1976b; Senay and Kok, 1977). This adaptation of body fluid systems to exercise training appears to be universal in mammals (McKeever *et al.*, 1985; McKeever *et al.*, 1986) and is exaggerated by exercise training in the heat (Senay, 1979).

The hypervolemic state induced by endurance training results in many physiological responses during exercise similar to those observed following acute hyperhydration. However, in contrast to the limitations of increasing body fluids by acute drinking, the elevation in plasma volume with endurance training represents just a small fraction of the total increase in total body water from 45.0 to 47.5 liters (Convertino and Kirby, 1984b). This water distribution provides greater interstitial fluid for sweating and thermoregulatory stability, as well as larger vascular fluid volume for cardiovascular stability. The mechanisms associated with this chronic expansion of body fluids and electrolytes include repeated acute elevations of plasma vasopressin and renin-angiotensin-aldosterone (Convertino *et al.*, 1980a; Convertino *et al.*, 1980b) as well as a chronic increase in total circulating protein (Convertino *et al.*, 1980b; Senay *et al.*, 1976b).

Consequent to the hyperhydration effect of exercise training is an alteration in the dynamics of fluid shifts during acute long-term exercise. At the same absolute exercise intensity, the percent plasm shift is reduced in the trained state (Convertino *et al.*, 1983b; Pivarnik and Senay, 1986). However, because the resting plasma volume is elevated, the absolute amount of plasma shifted out of the vascular space is similar to, or more than that observed in the non-trained state; furthermore, the plasma volume remaining in the vascular space is greater in the trained state (Convertino *et al.*, 1983b). At the same relative exercise intensity, i.e., percent of maximal oxygen uptake, trained and untrained individuals have similar percent plasma shifts, which represent a greater absolute amount of fluid efflux to the interstitial spaces for the trained individual. These responses are closely associated with lower levels of plasma renin-angiotensin and vasopressin during exercise at equal exercise intensities (Convertino *et al.*, 1983b). Similar to the comparisons between hyperhydration and dehydration states, greater vascular volume and fluid shifts during exercise in the trained state are highly correlated with greater sweat rate and lower heart rate at similar exercise intensities (Convertino *et al.*, 1983a; Senay *et al.*, 1976b).

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